"MYOCARDIAL PERFORMANCE INDEX AS A PREDICTOR OF ANGIOGRAPHIC SEVERITY OF CORONARY ARTERY DISEASE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION"

Dissertation Submitted

In partial fulfillment of the regulations

For the award of the degree of

DM BRANCH-II

CARDIOLOGY

STANLEY MEDICAL COLLEGE, CHENNAI.

AUGUST 2007

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI.

CERTIFICATE

This is to certify that the dissertation entitled **"MYOCARDIAL PERFORMANCE INDEX AS A PREDICTOR OF ANGIOGRAPHIC SEVERITY OF CORONARY ARTERY DISEASE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION"** is the bonafide original work of Dr.S.SOCRATES, in partial fulfillment of the requirements for D. M. Branch-II (CARDIOLOGY) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in August 2007.

Professor Dr.R.Subramanian, MD, DM.,
Professor and Head
Department of Cardiology
Government Stanley Medical College
& Hospital, Chennai.

DEAN
GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL
CHENNAI.

ACKNOWLEDGEMENT

At the outset, I wish to express my respect and sincere gratitude to my beloved teacher **Prof.R.Subramanian**, **M.D., D.M., (Cardiology) Professor & HOD, Department of Cardiology**, for his valuable guidance and encouragement through out the study.

I am extremely thankful to our **Additional Professor Dr.M.Somasundaram, M.D., D.M (Cardiology)** for his support and guidance during the study.

I am also expressing my thanks to all our **Assistant Professors of Cardiology** for their support during the study.

I thank the **Dean, Dr.T.Raveendran, M.D. (Chest), DTCD.** Government Stanley Medical College, Chennai for permitting me to utilize the hospital materials for conducting this study.

I express my thanks to **Mr.A.Venkatesan, Lecturer in Statistics,** clinical epidemiology unit, Government Stanley Medical College for his help in statistical analysis.

Last but not the least, I thank all the patients and controls who ungrudgingly lent themselves to undergo this study without whom this study would not have seen the light of the day.

CONTENTS

1.	**INTRODUCTION**	**1**
2.	**AIM OF THE STUDY**	**3**
3.	**REVIEW OF LITERATURE**	**4**
4.	**MATERIAL AND METHODS**	**50**
5.	**RESULTS**	**53**
6.	**DISCUSSION**	**58**
7.	**CONCLUSION**	**64**
8.	**BIBLIOGRAPHY**	**65**

INTRODUCTION

Cardiovascular disease is the second most common cause of death after communicable diseases in India. Coronary heart diseases accounts for more than 50% of cardiovascular disease deaths. STEMI occurrence is a fatal event in approximately 20 to 30% of patients. Nearly, one third of death occurs within 1 hour are mainly due to ventricular arrhythmias. But the late mortality is mainly depended on LV function. Hence LV function assessment is an integral part of any patient with acute STEMI.

While several methods are available for assessing LV function, Echocardiograph is the most readily available and commonly used method for the assessment of LV function. LV function derangement can affect systolic function, diastolic function or both. Two – dimensional echocardiography is well suited for studies of systolic function, and Doppler echocardiography provides a noninvasive technique for the assessment of diastolic function. However these measurements are load-dependent and change with the location of the sample volume, rhythm, heart rate and quality of the image. Even though systolic and diastolic dysfunction often coexists, only a few Doppler echocardiography variables combine measurements of systolic and diastolic performance. Recently, a new echocardiographic index combining the measurements of diastolic and systolic performance was defined. It was proposed first by Tei et al. In its short life span, it has been shown to demonstrate powerful prognostic value in significant heart diseases such as Dilated cardiomyopathy, Idiopathic pulmonary hypertension, Amyloidosis and recently in Myocardial infarction. TEI index has also got various other advantages compared to classical 2D and Doppler parameters such as, not influenced by change in blood pressure, heart rate, age and not appearing to be affected significantly by loading conditions. In our study its correlation with angiographic severity of coronary artery disease is assessed.

AIM OF THE STUDY

- To evaluate whether Myocardial performance index predicts Angiographic severity of coronary artery disease, and to assess the relationship between Myocardial performance index and Systolic, diastolic dysfunction in Acute STEMI patients.

MYOCARDIAL INFARCTION -AN OVER VIEW

Despite impressive improvements in diagnosis and management over the last four decades STEMI continue to be a major public health problem in the industrialized as well as developing economics like India. Our country is in the midst of epidemic of Diabetes and its resultant cardiovascular complications mainly due to improving socioeconomic status, westernization, changing lifestyles, obesity, stress and high incidence of insulin resistance seen in our population.

The diagnosis of Myocardial infarction requires presence of at least two of the following; characteristic symptoms, electrocardiographic changes and a typical rise and fall in biochemical markers. The pathological hallmark of Myocardial infarction is coronary atherosclerosis with superimposed occlusive thrombus.

Identification and management of risk factors are essential for preventing coronary heart disease in a symptomatic individual and for preventing recurrent events in patients with established disease. The efficacy of secondary prevention of CHD has been well established and this generated enthusiasm for extending the same to primary prevention. The key parameter for risk assessment is defining the absolute risk, i.e., the probability of developing CHD over a time period.

Absolute risk can be divided in to high, intermediate, and low risk categories based on 10 year absolute risk of developing myocardial infarction as follows, High (> 20%),

Intermediate (10-20%), Low (<10%) risk. High-risk patients are considered as coronary artery disease equivalent and it includes patient with non-coronary form of clinical atherosclerotic disease, type 2 diabetes mellitus, and symptomatic patients with presence of multiple risk factors other than DM.

ASSESSMENT OF NORMAL CARDIAC FUNCTION

The study of cardiac function has progressed from a description of cardiac anatomy to quantifying physiology and to the unraveling of the molecular pathways. Assessment of cardiac function is necessary for determining the diagnosis, for prognostication, timing of intervention, assessment of therapy, detecting complication and for assessing clinical outcomes.

The following 3 conditions must be met to consider cardiac function is entirely normal,

1) Normal systolic function. 2) Normal myocardial relaxation.

3) Normal diastolic filling pressures at rest and with exertion.

LEFT VENTRICUAR SYSTOLIC FUNCTION

The fundamental task of the cardiovascular system is to supply adequate quantities of oxygenated blood to the peripheral tissue. An important determinant of cardiac performance is left ventricular systolic function, which is determined by preload, after load, myocardial contractility and heart rate.

The surrogate markers for the preload are ventricular end-diastolic volume, end-diastolic diameter and end diastolic pressure. Quantifying after load in the intact circulation is more

challenging one. Two approaches are used, one focuses on the vascular load, which is measured by peripheral vascular resistance. The next approach focuses on the development of tension in the ventricular wall. Contractility manifest in the intact circulation at the rate of pressure development and of shortening from any given preload. Measures of systolic function and contractility are often considered together and include stroke volume, ejection fraction, the maximum rate of pressure increases during isovolumic contraction, and a variety of more sophisticated measurements such as end systolic pressure volume relationship, velocity of circumferential fiber shortening (VCF), afterload corrected (VCF).

Clinical parameters for assessing left ventricular functioning include the following[1],

1. Cardiac index (litre/min/m^2); heart rate × stroke volume per body surface area
2. Stroke volume index
3. Stroke work index; stroke volume index × mean systolic blood pressure.
4. Stroke force index; stroke work index per ejection period in seconds.
5. Pre load recruitable stroke work = relationship between stroke work and end diastolic volume.

There are several indices of global left ventricular systolic function and contractility. Each index is variably dependent on preload and after load and can be modified by ventricular volume and myocardial mass. The ease of application to the clinical setting is an important feature.

DIASTOLIC FUNCTION

Diastolic function has been found to play an important role in cardiac morbidity and mortality and to influence both preload and after load. From a clinical standpoint, four separate

phases of diastole need to be distinguished: 1) Isovolumic relaxation 2) Early diastolic filling 3) Slow ventricular filling (diastasis) 4) Atrial filling.

Diastolic function is influenced by several factors e.g. myocardial relaxation, ventricular filling, and the ventricle's passive elastic properties, but one of the major determinants is heart rate, which determines how much time is available for ventricular filling. An increase in heart rate shortens the diastolic filling time interval disproportionately. This reduction must be compensated by an increase in the rate of relaxation and an augmentation of elastic recoil with enhanced diastolic suction.

In the normal left ventricle, the end–systolic volume is smaller than its elastic equilibrium and it thus generates elastic recoil, which varies inversely with the end-systolic volume. The elastic recoil causes diastolic suction that fills the ventricle at a low pressure and induces a potential for –ve left ventricular pressure in early diastole. This filling mechanism is important during exercise and allows the normal ventricle to reduce minimal diastolic pressure and to maintain end diastolic pressure constant despite a 3 fold to 5 fold increase in cardiac output. A loss of elastic recoil occurs during acute ischemia with reduction in early diastolic filling accompanied by an increase in left atrial filling pressure and heart rate.

Another important determinant of diastolic function is the atrioventricular pressure gradient, which is dependent on atrial pressure, relaxation rate, viscous forces in the myocardium, and ventricular filling rates.

ASSESSMENT OF SYSTOLIC FUNCTION

M - MODE ECHOCARDIOGRAPHY

Measurement of the LV cavity dimension and wall thickness can be readily derived from M mode recordings and are usually made according to the recommendation of the American society of Echocardiography at end diastole and end systole. These measurements should be made from leading edge to leading edge to avoid incorporating artifacts and reverberation.

The M-mode LV cavity dimension can be used to estimate **ventricular volumes** and **ejection fraction** if desired, most simply by merely cubing (**teicholtz**) the value. But this calculation involves several assumptions regarding LV geometry that are not uniformly valid. In addition, M mode dimension may not be representative of the entire ventricle. The **fractional shortening** can also be measured. This value is often helpful in assessing systolic function, but it reflects the function of LV in one chord and in one plane and can be misleading with asynchronous contraction (for example LBBB) or segmental dyssynergy. An additional M-mode marker of systolic dysfunction is E point septal separation **(EPSS**). A value of 8 mm or greater is abnormal.

2D ECHOCARDIOGRAPHY

Since 2D-echocardiography enables visualization of the entire LV perimeter in multiple planes, it is significantly superior to M- mode approaches for the measurement of cardiac chamber volumes and EF. Numerous algorithms have been applied to calculate LV volumes by

echocardiography. Most such algorithms have assumed that the LV conforms to the shape of a prolate ellipsoid and calculated volume by **diameter - length** or **area - length** formulas. Multiple studies comparing LV volume calculated by area length method to those obtained by other techniques have yielded good correlation, with best results obtained utilizing biplane apical views. Currently the most commonly used algorithms to calculate LV volume is based upon the **SIMPSON** rule, which derives measurements by dividing the LV by parallel planes into a number of small segments and then summating the area of the individual disks. This approach makes less assumption about the geometry of the ventricle. Several modifications of the basic Simpson rule method have been applied to calculate LV volumes. The optimal correlation has been achieved with a modification that separately quantifies the volume of the apex as an ellipsoid.

Accurate calculation of LV volumes by Echocardiography requires high quality images to delineate the endocardium and image the entire LV perimeter. End systolic measurements are more accurate than those made at end diastole, probably owing to the superior endocardial definition. Nevertheless, Echocardiography calculations of LV volumes have generally yielded correlation coefficients in excess of 0.75 as compared with radionuclide angiography, cineangiography and autopsy studies. Hence calculations of LV volumes by these measurements are suitable for clinical decision making in the care of most patients.

DOPPLER ECHOCARDIOGRAPHY

Although measurements of LV volume and ejection fraction can be obtained by 2D Echocardiography, Doppler interrogation provides the unique and complimentary Non-invasive

assessment of systolic function. Thus LV systolic dysfunction often results in decreased aortic velocity and acceleration time. In the presence of mitral regurgitation, the acceleration of the MR jet can provide information regarding contractile function.

One of the most important applications of Doppler is in the calculation of the stroke volume. The volume of flow through any orifice or tube can be calculated as the product of cross - sectional area through which flow occurs and the velocity of that flow. Measurement of cross sectional area can be derived from Echocardiography images, while velocity can be determined by Doppler. The cardiac output can be derived from the product of stroke volume and heart rate. Although the calculation of stroke volume by the Doppler method involves number of assumption, it has been shown to correspond well with thermo dilution, Fick, and the angiography calculations.

REGIONAL FUNCTION

LV regional wall motion analysis is usually based on grading of contractility of individual segments. There are various LV segmental models depending of how the LV is subdivided. For the purpose of standardized analysis, the LV is divided into three levels (basal, mid, and apical) and 16 segments. The basal and mid (papillary muscle) levels are each subdivided into 6 segments, and the apical level is subdivided into 4 segments. Recently a 17 segments approach has been recommended[2], in which the 17th segment represents true apex. In general the anterior septum, anterior wall are perfused by the LAD artery and its branches, the inferior wall in the area of posterior interventricular groove by the RCA. There can be substantial overlap in the inferior, lateral and anterolateral segments, depending on the dominance of the RCA and LCX coronary arteries. The inferoapical segment represents an overlap zone between the distal LAD and distal RCA, and the apical lateral wall represents an overlap between the LCX and LAD arteries.

A ventricle with a normal wall motion would have a score index of 1.0 (total score

divided by the number of segments), with higher scores representing progressively greater degrees of ventricular dysfunction. This score can be sub divided into **anterior score**, representing the distribution of LAD and a **posterior score** representing the RCA and LCX territories. Patients with WMSI greater than 1.7 had a perfusion defect larger than 20%. Based on the contractility of the individual segments, a numerical scoring system has been adopted in which higher scores indicate more severe wall motion abnormality (1 = normal; 2 = hypo kinesis; 3 = akinesis; 4=dyskinesis, 5 = aneurysmal). A wall motion score index (WMSI) is derived by dividing the sum of wall motion scores by the number of visualized segments, and represents the extent of regional wall motion abnormalities.

$$\text{WMSI} = \frac{\text{SUM OF WALL MOTION SCORES}}{\text{NO. OF VISUALIZED SEGMENTS}}$$

DIASTOLIC FILLING PROFILES

Diastole begins with myocardial relaxation at the end of systolic contraction (A2, aortic valve closure) and ventricular pressure starts to decline (isovolumic relaxation). When the left atrial pressure exceeds the LV pressure, the mitral valve opens to allow an early (E) rapid filling phase of the LV. The LV pressure continues to decline after the onset of E, but soon increases again with continuous ventricular filling equalizing with left atrial pressure, resulting in a period of diastasis. Another bolus of ventricular filling occurs with left atrial contraction (A) whose contribution to the total C.O. depends on LV filling pressure and atrial contractility.

Pulse-wave Doppler mitral flow velocities reflect the Trans mitral pressure changes

during diastole. Mitral flow velocities are measured by pulse-wave Doppler, with the sample volume placed between the leaflet tips, and the following diastolic filling parameters are derived: IVRT, early filling velocity (E), late filling velocity (A), and Deceleration Time (DT) of E. When the ventricular filling pattern is evaluated, it is important to keep the sample volume location. Both E and A velocities are smaller with the sample volume at the mitral annulus compared to the leaflet tip position. The DT is also shorter at the annulus position.

GRADING OF DIASTOLIC DYSFUNCTION[3]

Grade I - Impaired relaxation pattern with normal filling pressure.

Ia. - Impaired relaxation pattern with increased filling pressure.

2. - Pseudonormalisation pattern.

3. - Reversible restrictive pattern.

4. - Irreversible restrictive pattern.

NORMAL DIASTOLIC FILLING PATTERN:

The rates of myocardial relaxation and compliance change with aging, so that different diastolic filling patterns are expected for different age groups. In normal young subjects, LV elastic recoil is vigorous and myocardial relaxation is swift, therefore, most filling is completed during early diastole with only small contribution at atrial contraction. Therefore E/A ratio is usually 1.5 or higher, DT 160-230 mess, E' 10 cm/s or more; E/E' less than 8, VP 50 cm/s or more. With valsalva, both E and A velocities decrease with lengthening of DT, so that the E/A ratio remains the same.

With aging, the rate of myocardial relaxation and elastic recoil gradually decrease, resulting in slow LV pressure declines and slower filling. At the age of 65 years, E velocity approaches a velocity, and in persons older than 70 years, E/A ratio is usually less than 1.0. Pulmonary vein flow velocities show similar changes with aging.

ABNORMAL RELAXATION

When myocardial relaxation is the predominant diastolic abnormality, IVRT is prolonged and the initial decline in LV pressure is slow. So, early filling is reduced, and there is a large compensatory filling with atrial contraction. The ventricle continues to relax even after the opening of the mitral valve, and it takes longer to equalize ventricular pressure with atrial pressure, resulting in a longer DT.Therefore, abnormal myocardial relaxation is characterized by a constellation of abnormalities consisting of[4]

1. Prolonged IVRT (>90 mess)
2. PVS_2 >> PVd
3. E/A ratio (<1.0)
4. Prolonged DT (>240msec)
5. Mitral A duration ≥ (or) < PVa duration depending on LVEDP.

RESTRICTIVE PHYSIOLOGY

When ventricular compliance is decreased, the rise in ventricular diastolic pressure is very rapid during the early filling phase (short DT) and the elevated LV end –diastolic pressure minimizes ventricular filling due to atrial contraction (decreased A). With the resultant high left atrial pressure, the IVRT becomes shortened and the E velocity is high. This particular diastolic

filling pattern is indicative of "restrictive" physiology characterized by the following diastolic parameters[4]:

1. Shortened IVRT (< 70 msec)

2. High E velocity and low A velocity.

3. Increased E/A ratio (≥ 2.0)

4. Shortened DT (< 160 msec)

5. PVS_2 << PVd

6. Mitral A wave duration < PVa duration.

7. PVa velocity increased (≥ 35 cm/s, usually but not always). Restrictive physiology pattern is seen whenever LV diastolic pressure rises rapidly and end diastolic pressure is high, as in LV failure, restrictive cardiomyopathy, volume overload, and severe acute aortic regurgitation.

PSEUDO NORMALIZATION

When relaxation abnormality and restrictive hemodynamics coexist, Doppler features of the latter predominate. The pseudo normalization pattern is present when the left atrial pressure rises moderately in the setting of abnormal myocardial relaxation, producing a diastolic filling pattern similar to the normal pattern. Pulmonary venous systolic forward flow velocity is decreased in pseudo normalization, whereas it is higher than diastolic forward flow in the true normal filling pattern. Pulmonary venous flow velocity helps in separating pseudo normal from true normal diastolic filling. The elevated left atrial pressure in the patient with pseudo normal mitral inflow produces a longer duration and higher velocity of pulmonary vein atrial flow reversal. The pseudonormalisation pattern is characterized by,

1. DT 160-200 msec.

2. IVRT < 90 msec

3. E/A - 1-1.5

4. PVS2 < PVd

5. Mitral A duration < PVa duration

6. Pva Velocity > 35 cm/s.

VENOUS FLOW PATTERN IN DIASTOLIC FILLING ASSESMENT

Venous flow (pulmonary and hepatic vein) velocity patterns are also useful in the evaluation of diastolic filling of respective ventricle. Analysis of the **pulmonary venous filling patterns** provides a 2nd window into LV diastolic function. The S wave, occurring during systole depends on atrial relaxation, D wave occurring during diastole reflects left ventricular filling, and the A wave, which is opposite the other waves occur during atrial contraction, reflects left ventricular compliance. One indication for examining pulmonary venous flow pattern is to distinguish the truly normal filling from pseudo normalization. In the presence of pseudo normalization pattern atrium contracts against an increase in after load due to an elevated filling pressure of a stiff ventricle, hence blood is preferentially ejected in to pulmonary veins, resulting in a high and prolonged pulmonary venous a wave.

In normal subjects **Hepatic vein flow** velocities consists of four components, systolic forward flow (S), diastolic forward flow (D), systolic flow reversal (SR), diastolic flow reversal (DR). Under normal hemodynamic conditions, S is higher than D and there is no prominent reversal velocities. As RV filling pressure increases, the hepatic systolic flow velocity decreases and diastolic flow velocity increases, analogous to the pulmonary vein pattern. With

marked increase in RV filling pressure, a prominent flow reversal occurs during systole and diastole or both.

COLOUR M MODE DOPPLER ECHO CARDIOGRAPHY

A limitation to conventional pulse wave Doppler is that it only provides the velocity of blood flow at a single point within the heart. Colour M mode recordings overcome this limitation by providing the spatial and temporal velocity characteristics of flow along an entire echocardiographic scan line. Color M mode echo is a useful technique for examining the dynamics of blood flow across the mitral valve. The velocity of blood flow is increased with rapid relaxation and LV suction. Combined evaluation of flow propagation velocity and early diastolic annular velocity can be used for estimation of filling pressure.

TISSUE DOPPLER IMAGING

Tissue Doppler imaging refers to the technique of determining directional velocities of tissue structures rather than the moving blood pool. Tissue Doppler imaging yields information on intra myocardial velocity, providing a unique insight into LV mechanics during isovolumic contraction and relaxation. Doppler evaluation of annular motion has shown tremendous promise for the evaluation of diastolic function. When evaluated from an apical transducer position, annular motion is opposite in direction to the mitral inflow signals. The early annular velocity (Ea) exceeds late annular velocity (Aa) in a manner similar to mitral valve E/A. In patients with pseudo normal or restrictive filling pattern annular motion is abnormally low implying that it is relatively independent of pre-load. Doppler tissue imaging can be used to calculate direction and velocity of motion in two adjacent myocardial segments of known

distance of separation. From this strain, reflecting the relative velocity of either separation or closure between these two points can be calculated. This can further be developed into **"strain rate imaging"**, which integrates the rate of distance change between 2 adjacent points over time, which in turn is a parameter that can be color encoded over a segment of the myocardium. Both experimental animal and clinical data suggest that strain rate imaging can provide an increased level of resolution and accuracy for identification of subtle wall motion abnormalities in patients with ischemic and those with nonischemic heart disease. Through the integrated use of Doppler echo and tissue Doppler imaging, it is possible to obtain a fairly precise picture of left ventricular systolic and diastolic functions. However atrial fibrillation or frequent ectopic beats introduce major limitations of these techniques.

LV FUNCTION IN MYOCARDIAL INFARCTION

SYSTOLIC FUNCTION

When antegrade flow in an epicardial coronary artery is interrupted, the zone of myocardium supplied by that vessel immediately loses its ability to shorten and perform contractile work. Four abnormal contraction patterns develop in sequence; 1.**Dyssynchrony**-dissociation in the time course of contraction of adjacent segments, 2) **Hypokinesis**-reduction in the extent of shortening; 3) **Akinesis**-cessation of shortening and 4) **Dyskinesis**-paradoxical expansion and systolic bulging. Accompanying dysfunction of the infarcting segment initially is hyper kinesis of the remaining normal myocardium. The early hyperkinesis of the non-infarcted zones is thought to be the result of acute compensatory mechanisms, including increased activity of the sympathetic nervous system and the Frank-starling mechanism.

Increased motion of the noninfarcted region subsides within 2 weeks of infarction, during which time some degree of recovery can be seen in the infarct region as well, particularly if reperfusion of the infracted area occurs and myocardial stunning diminishes. If a sufficient quantity of myocardium undergoes ischemic injury, left ventricular pump function become depressed, cardiac output, stroke volume, BP, and peak dP/dt are reduced and end systolic volume is increased. The degree to which end systolic volume increases is perhaps the powerful predictor of mortality following STEMI.As the ventricle dilates during the first few hours to days after infarction, regional and global wall stress increase according to Laplace's law. The degree of ventricular dilation, which depends closely on infarct size, patency of the infract related artery, and activation of the local rennin- angiotensin system in the noninfarcted portion of the ventricle can be favorably modified by angiotensin converting enzyme (ACE) inhibition therapy, even in the absence of symptomatic left ventricular dysfunction.

The earliest abnormality is a reduction in diastolic compliance, which can be observed with infarcts that involves only 8 % of the total left ventricle on angiographic examination, when the abnormally contracting segment exceeds 15%, the ejection fraction may be reduced and elevations of left ventricular end diastolic pressure and volume occur. The risk of developing physical signs and symptoms of left ventricular failure also increase proportionally to increasing areas of abnormal Left Ventricular wall motion. Clinical heart failure accompanies areas of abnormal contraction exceeding 25% and cardiogenic shock, accompanies loss of more than 40%of the left ventricular myocardium. Unless infarct extension occurs, some improvement in wall motion takes place during the healing phase, as recovery of function occurs in initially reversibly injured (stunned) myocardium. Regardless of the age of

the infarct, patient who continues to demonstrate abnormal motion of 20 to 25%of the left ventricle are likely to manifest hemodynamic signs of LV failure.

DIASTOLIC FUNCTION

The diastolic properties of the left ventricle are altered in infarcted and ischemic myocardium. These changes are associated with a decrease in the peak rate of decline in left ventricular pressure [peak (-) dP/dt], an increase in the time constant of the fall in left ventricular pressure, and an initial rise in left ventricular end-diastolic pressure. Over a period of several weeks, end-diastolic volume increases and diastolic pressure begins to fall toward normal. As with impairment of systolic function, the magnitude of the diastolic abnormality appears to be related to the size of the infarct.

WALL MOTION ABNORMALITIES IN ACUTE CORONARY SYNDROMES

Experimentally, immediately after coronary artery occlusion, abnormalities in diastolic function occur. The easiest and most commonly identified abnormality is abnormal mitral valve inflow, with reduction in E wave velocity and an increase in A wave velocity occuring within seconds of total coronary occlusion. Depending on the presence and absence of collaterals and the duration of occlusion, a series of identifiable wall motion abnormalities can be noted. If the coronary artery obstruction persists for a threshold period (typically defined as ≥ 4 hours), myocardial necrosis ensues and a persistent wall motion abnormality will develop.

In most instances, a total occlusion of 4-6 hours will result in irreversible loss of transmural myocardium. Total interruption for less than 60mins will results in lesser degrees of

loss of myocardium. In between these two extremes of flow interruption, varying degrees of non-transmural necrosis occur.

Myocardial stunning represents persistent wall motion abnormalities after restitution of coronary blood flow. These abnormalities will recover over a variable period of time typically, with brief occlusions of 5 min or less, recovery of function occurs within 60-120 seconds. With coronary occlusions of 30-120 min, there may be a 48-72 hours delay in recovery of function. There is a substantial degree of variability in the time course over which stunning recovers, and occasionally is delayed for weeks to months.

The extent of wall motion abnormality often overestimates the anatomic extent of ischaemic or infarction. This is in large part due to tethering. **Myocardial tethering** refers to the impact that an abnormal segment has on a normal adjacent border segment. Tethering occurs both on a horizontal and vertical basis. **Horizontal tethering** results in the reduction in endocardial excursion in the adjacent functionally normal boundary tissue. **Vertical tethering** refers to contraction abnormality in the subendocardial region having a disproportionate impact on overall wall thickening. In general, ischemia or infarction of the inner 20% of the myocardial wall will result in frank akinesis or dyskinesis of that segment.

The **current role of Echocardiography in Acute Myocardial Infarction** can be classified as follows[4]:

1. Diagnosis and evaluation of Acute Myocardial Infarction in patients with prolonged chest pain and non-diagnostic ECG findings.

2. Estimation of the amount of myocardium at risk and final infarct size after reperfusion therapy.
3. Evaluation of unstable haemodynamics.
4. Detection of infarct complications.
5. Evaluation of myocardial viability.
6. Risk stratification.

ANGIOGRAPHIC ASSESSMENT OF CORONARY ARTERY NARROWINGS

An angiographic lumen narrowing is commonly referred to as a stenosis, which may be due to atherosclerosis, vasospasm, or angiographic artifact.

To quantify the coronary stenosis accurately it must be seen in profile, free from artifact related to foreshortening or obfuscation by a crossing vessel. Multiple views are important, because many lesions have a markedly eccentric lumen. When seen across its major axis, the width of the lumen may appear to be normal, but a clue to the presence of a severe degree of narrowing in the other axis may be marked lucency caused by thinning of the contrast column. Any such suspicious lesions must be examined in a variety of other projections.

The normal caliber of major coronary arteries, Left main 4.5±0.5mm,LAD 3.7±0.4mm, LCX 3.4±0.5mm for non-dominant and dominant LCX 4.2 ±0.6mm; RCA 3.9 ± 0.6 for dominant and 2.8 ± 0.5 for non dominant[5].

By comparing the diameter of a presumably disease free segment of coronary artery to the size of the diagnostic catheter (6F equals 2mm), the operator can identify vessels that fall below these normal size ranges and may thus be diffusely diseased. The evaluation stenosis relates to the percentage of reduction in the diameter of narrowed vessel site to the adjacent unobstructed vessel. The diameter stenosis is calculated in the projection where the greatest narrowing in seen. It should be noted that the stenotic lumen is compared to near by unobstructed lumen, which indeed may have diffuse atherosclerotic disease and thus is angiographically normal but still may be diseased. The **area of stenosis is always greater than diameter stenosis** and assumes the lumen is circular when in reality most of the time the lumen is eccentric.

In 1975 American Heart Association recommended that the diameter method be adopted for grading coronary artery stenosis[6]. Six categories of coronary narrowing have been commonly used:

1. Normal coronary artery
2. Irregularities of the vessel
3. Narrowing of less than50%
4. Stenosis between 50 and75%
5. Stenosis between 75 & 95%
6. Total occlusion

A 50 % of reduction in diameter is equivalent to a 75% reduction in Cross Sectional

area, and a 75% reduction diameter is equal to a 90 % reduction in cross sectional area. Stenosis that reduces the lumen diameter by 50% (and hence cross sectional area by 75%) is hemodynamically significant, in that it reduces the normal three fold to four fold flow reserve of coronary bed. Whereas a diameter stenosis of 70% (90% cross sectional area) eliminates virtually any ability to increase flow above it's resting level. Stenosis that reduced the lumen diameter by 90%, however, rarely exists without reducing antegrade flow[7]. Because of the subjective nature of a visual lesion assessment, there is a ± 20% variation between readings of two or more experienced angiographers especially for lesions 40 – 70% narrowed.

The simplest way to resolve this problem is, project the coronary image on a wall mounted viewing screen, and use inexpensive digital calipers to measure the relative diameters of the stenotic and reference segment. Percent stenosis can be calculated as 100 × (1 – stenosis diameter/reference diameter) to provide a more accurate estimate of stenosis.

ANGIOGRAPHICALLY ESTIMATED CORONARY BLOOD FLOW (TIMI FLOW)

Myocardial blood flow has been assessed angiographically using Thrombolysis in Myocardial infarction score for qualitative grading of coronary flow. TIMI flow grades 0 – 3 have become a standard description of angiographic coronary blood flow in clinical trails[8]. In acute myocardial infarction trails TIMI grade 3 flow have been associated improved clinical outcomes. The four grades of flow are described as follows:

TIMI 3 (complete reperfusion) – Anterograde flow into the terminal coronary artery segment through a stenosis is as prompt as Antegrade flow into a comparable segment proximal to

stenosis. Contrast material clears as rapidly from the distal segment as from an uninvolved, more proximal segment.

TIMI 2 (partial reperfusion) - Contrast material flow through the stenosis to opacify the terminal artery segment. However, contrast material enters the terminal segment perceptibly more slowly then more proximal segments. Alternatively contrast material clears from a segment distal to a stenosis noticeably more slowly than from a comparable segment not preceded by a significant stenosis.

TIMI 1 (penetration with minimal perfusion) - A small amount of contrast material flows through the stenosis but fails to opacify fully the artery beyond.

TIMI 0: (No perfusion) – No contrast flow through the stenosis.

Quantitative method of TIMI uses cine angiography with 6F catheters and filming at 30 frames per second. The number of cine frames from the introduction of dye in the coronary artery to predetermined distal landmark is counted.

The first frame used for TIMI frame counting is that in which the dye fully opacifies the artery origin and in which the dye extends across the width of the artery touching both borders with anterograde motion of the dye. The last frame counted is when the dye enters the first landmark branch. Full opacification of distal branch not required. The distal landmarks commonly used in analyses are,

(1) for LAD -the distal bifurcation of LAD.

(2) for the circumflex system, the distal bifurcation of branch segments with the longest total distance.

(3) for the RCA the first branch of posterolateral artery.

The TIMI frame count can further be quantified for the length of the LAD for comparison to the two other major arteries. This is called **corrected TIMI frame count (CTFC)**[9]. The average LAD coronary artery is 14.7cm long, RCA 9.8cm, LCX 9.3cm. CTFC accounts for the distance the dye has to travel in the LAD relative to the other arteries. CTFC divides the absolute frame count in LAD by 1.7 to standardise the distance of dye travel in all the three arteries. Normal TFC for LAD is 36±3 and CTFC 21±2, for LCX TFC is 22±4, for RCA TFC is 20±3. TIMI flow grades do not correspond to measured Doppler flow velocity or CTFC. High TFC may be associated with micro vascular dysfunction despite an open artery. CTFC of <20 frames were associated with low risk for adverse events in patients following myocardial infarction. A contrast injection rate of >1ml/sec by hand injection can decrease the TIMI frame count by 2 frames. The TIMI frame count method provides valuable information relative to clinical responses after coronary intervention.

COLLATERAL CIRCULATION

The reopacification of a totally or sub totally (99%) occluded vessel from antegrade or retrograde filling is defined as collateral filling. In the normal human heart, network of tiny anastomotic branches interconnect the major coronary arteries and are precursors for the development of a collateral circulation[10]. These anastomotic arteries cannot be visualized in patients with normal or mildly diseased coronary arteries because they carry only minimal flow and their small (200µm) caliber is well beyond the spatial resolution capabilities of coronary imaging systems. As the obstruction progresses in the coronary arteries, a pressure gradient is generated within the anastomotic vessels connecting the distal hypoperfused segment with the proximal artery or the adjacent anastomotic channels of other vessels. The transstenosis

pressure gradient facilitates blood flow through the anastomotic vessels, which progressively dilates and eventually become visible as collateral channels. Angiographically visible collateral channels are not usually seen until the coronary obstruction is greater than 90%, at which point coronary perfusion pressure falls substantially and the blood flow through the collaterals increases.

Collaterals arise either from the contralateral coronary artery, called **inter coronary collaterals** or from the ipsilateral coronary arteries through **intra coronary collaterals** or through **bridging channels** that have a serpiginous course from the proximal coronary artery to the coronary artery distal to the obstruction. The collateral circulation may provide upto 50% of anterograde coronary flow in chronic total occlusions.

RENTROP COLLATERAL GRADE:

Grade 3: Complete perfusion, contrast materials enters and completely opacifies the target epicardial vessels.

Grade 2: Partial collateral flow, contrast material enters but fails to opacify the target epicardial vessel completely.

Grade 1: Barely detectable collateral flow, contrast medium passes through collateral channel but fails to opacify the epicardial vessel any time.

Grade 0: No collaterals present.

Among the patients studied within the 6hrs of STEMI, about half show angiographically visible collaterals. When the angiography was done more than 24hrs after STEMI, virtually all patients had visible collaterals. Incidence of collaterals 1-2weeks after STEMI varies considerably and may be as high as 75-100 % in patients with persistent occlusion of the infarct vessel or as low as 17-42 % in patients with subtotal occlusion.

CORONARY ANATOMY AND LOCATION OF INFARCTION

In more than 75%patients with STEMI who came to autopsy, more than one coronary artery is severely narrowed. Approximately one half of the patients with STEMI have critical obstruction (more than 75 % of luminal area obstructed) of all 3 coronary arteries. The reminder is equally divided between one vessel and two-vessel disease.

Coronary angiographic study in surviving patients of STEMI shows, a higher percentage has single vessel disease. Angiographic studies performed in the earliest hours of STEMI have revealed approximately a 90% incidence of total occlusion in the infarct related vessel[11]. Recanalisation from spontaneous thrombolysis as well as attrition due to some mortality among those patients with total occlusion results in diminishing incidence of total occluded vessel in the period following the onset of myocardial infarction.

A STEMI with transmural necrosis occurs distal to an acutely occluded coronary artery with thrombus superimposed on a ruptured plaque. However, chronic total occlusion may not be associated with MI. Collateral blood flow and other factors such as the level of myocardial metabolism, the presence and location of stenosis in other coronary arteries, rate of development of the obstruction and quantity of the myocardium supplied by the obstructed vessel, all influence the viability of cells distal to the occlusion.

In about 5%patients with STEMI (studied by angiographically or in autopsy series) found to have normal coronary vessels. In these patients, an embolus that has lysed, a transiently occlusive platelet aggregate, or a prolonged episode of severe coronary vasospasm

may have been responsible for the reduction in coronary flow.

INFARCT RELATED ARTERY-PATENCY AND REOCCLUSION

In the early 1940's the possible relationship between a patent infarct related artery, myocardial damage, and mortality was first proposed. This theory is called **open vessel hypothesis**, holds that, early reperfusion results in myocardial salvage, preserves ventricular performance, and is ultimately responsible for improved patient survival.

The **clinical markers of coronary reperfusion** are[12],

(1) Pain relief - abrupt resolution of chest pain or >50%reduction in intensity at 90mts.

(2) More than 50% decrease in, pretreatment maximum ST elevation at 90mts of reperfusion therapy

(3) Appearance of T inversion more than 0.5mm below baseline within 24hrs of thrombolytic therapy in all the corresponding infarct related ECG leads with previous ST elevation

(4) Elevation of CK values in a proportion of more than 60%with respect to its maximum value before 12hrs of starting thrombolytic therapy.

GUSTO I and GUSTO angiographic study has provided **Predictors of infarct related artery patency** at 90mts-

1) Use of tPA (versus SK)

2) Body weight <85kg

3) History of smoking - current smokers achieve more patency of infarct related artery

4) Infarct related artery- proximal LCX and proximal RCA lesion, chance for patency

more.

Reocclusion of infarct related artery (TIMI 4 TRAIL)

Angiographic predictors of reocclusion of IRA at 90mts are

1) Presence of TIMI 2 flow (10.4% vs 2.2% in TIMI 3)

2) Lesion ulceration (10.7% vs 3% without ulceration)

3) Presence of collaterals (18.2% vs5.6% with out collaterals)

4) Lesions that are eccentric.

THE MYOCARDIAL PERFORMANCE INDEX (Tei INDEX)

There are many limitations to the use of classical Echocardiographic indexes for the estimation of systolic and diastolic LV function. The ejection fraction and LV Volumes are subject to large errors when the ellipsoid shape of the heart becomes spherical. Age, rhythm and conduction disturbances and changes in loading all affect the Doppler Signal of transmitral flow, which is the most commonly used method for studying diastolic function.

In the 1960's the duration of pre-ejection period and LV ejection time were studied extensively as a measure of cardiac systolic function and LV stroke volume. These indices were measured using phonocardiogram and carotid pulse tracings. In 1968, **Weissler etal**, derived an Index (Pre-ejection period / LVEF) called **systolic time interval,** which depends less on heart rate as a measure of LV Systolic function[13]. Because isovolumeic relaxation time is also affected by LV function, **Mancini etal**, in 1982 incorporated IVRT into an index called **isovolumic index**, derived as (IVCT + IVRT)/LVET. The sum of IVCT and IVRT was determined by subtracting LVET from the peak of the R wave in ECG to onset of mitral valve opening[14]. However the interval from the peak of the R wave to onset of mitral valve opening contains an interval of electro mechanical delay, which can be pronounced in patients with LBBB. With the advent of Doppler echocardiography, it has become easier to determine cardiac time intervals more reliably.

In 1995, **Tei Chuwa** devised and published an index of myocardial performance called Tei index, which evaluates the LV Systolic and diastolic function in combination[15].

The Tei index is a pure number and is calculated from the ratio of time intervals (a-b) / b derived with the aid of pulsed Doppler Echocardiography. Locating the sample volume at the tips of the mitral valve leaflets, in the apical 4-chamber view, enables the measurement of **a,** which is the time interval between the end and start of transmitral flow. The sample volume is then located in the LV outflow tract, just below the aortic valve (apical 5 chamber view) for the measurement of **b**, the LV ejection time. The interval **a** includes the isovolumic contraction time (IVCT), Ejection time (ET) and isovolumic relaxation time (IVRT), and the Tei index may also be expressed by the formula **(IVCT + IVRT) / ET**. For the evaluation of Right Ventricular Tei Index, the **a** interval, from the end to start of trans-tricuspid flow (the interval from the end of A wave to the Start of E wave), is obtained from the apical 4 chamber view with the Doppler sample volume located between the tips of the tricuspid valve leaflets. The interval (RVET) is measured from para sternal short axis view, with sample volume located just below the pulmonary valve. Normal Range of LV Tei index in an adult is **0.39 ± 0.05** and for RV is **0.28 ± 0.04**.

MODIFIED TEI INDEX

The major limitation of TEI index is that, it is not measured in a single cycle, rather the measurements are obtained sequentially, and hence in the presence of marked heart rate fluctuations reliability of this method is restricted. Hence a modification is proposed for measuring TEI index by using tissue Doppler.[16] This modification enables to measure contraction and relaxation velocities in a single cycle simultaneously. Modified TEI index is measured as follows; The TDI program is set to pulse wave Doppler mode, filters were set to exclude high frequency signals & gains were minimized to allow a clear tissue signal. The spectral Doppler signal is adjusted to a nyquistic limit of 15-20 cm/sec. TDI is obtained from apical 4chamber view & a 2 mm of sample volume is placed either at medial or lateral corner of mitral annulus. Velocities are recorded at sweep speed of 100 mm/sec. The time duration from end to onset of mitral annular velocity pattern during diastole is measured (measurement a'). The duration of S wave is measured from its onset to the end & it is marked as measurement **b'**. IVRT is calculated from subtracting the interval **d'** (R wave & cessation of systolic velocity), from the interval **c'** (R wave to onset of diastolic velocity). IVCT is calculated by subtracting IVRT from (a'-b'). Modified TEI INDEX by TDI is calculated as a'-b'/b'.

Tei Index and age

In a study[17] conducted in 161 children with no cardiovascular disease, aged from 30 days to 18 years, for determining the range of normal values for the Tei index and the effect of age. It was found that Tei index was affected by age during the first 3 years of life, showing a progressive reduction until the age of 3, but then it showed no further changes. The Tei index for children aged < 3 years was significantly greater (0.40 ± 0.09) than for those aged between3 and 18 (0.33 ± 0.02). The age-dependent changes in the index may reflect changes during the maturation of the myocardial characteristics of the LV in neonates and children. During development, the relation between total collagen and total protein reaches normal levels in 5 months and the relation between type I collagen (which mainly provides rigidly) to type III collagen (which provides elasticity) stabilizes after 3 years, The RV Tei index in 150 healthy children, mean age 5.1 ± 5.5 years, was 0.24 ± 0.04, irrespective of age.

Tei Index and preload

The effect of preload changes on the Tei index was investigated in 50 healthy volunteers and 25 patients with a previous infarction[18]. Three procedures were performed successively, the Valsalva maneuvers (preload reduction), passive rising of the lower limb (preload increase) and administration of sublingual nitroglycerine (preload reduction). In the controls, the index increased significantly during the Valsalva maneuver (mainly as a result of a reduction in ET), after passive rising of the lower limb (primarily as a consequence of an increase in IVCT) and after nitroglycerine administration (as a result of a reduction in ET and a prolongation of IVCT). In contrast, no significant changes were seen in the index in the infarction patients

during the above preload variations.

Briefly, in those patients when the preload decreased the IVCT/ ET ratio showed a reduction while the IVRT / ET ratio increased, leaving the index unchanged. Although these result show a change in the Tei index under different preload conditions, the extent of the changes was small (<10%), a fact that explains the preservation of the prognostic value of the Tei index despite variations in preload.

Tei Index and Afterload

In a population based study from Department of Geriatrics[19], Sweden, 446 men aged 70 years, MPI, LV mass, LVOT diameter, Time velocity integral of LVOT were measured. Stroke volume, cardiac index were calculated from these parameters. Total peripheral resistance index was obtained by formula: 80 x (MAP - 3) / cardiac index. Total arterial compliance was calculated by dividing stroke volume by pulse pressure. 24 hours ambulatory BP and HR were monitored. Both total peripheral resistance index and stroke volume/pulse pressure ratio are the variables reflecting afterload properties, as both resistance and compliance in the arterial system oppose the ejected blood volume. Both these variables were independently related to MPI. A high afterload would reduce the ejection time and thereby has profound consequences on MPI.

Tei Index and Haemodynamic parameters

In a prospective study 17 patients with idiopathic dilated cardiomyopathy (EF: 24% ± 11%) and 19 patients with ischaemic heart disease (EF: 49% ± 13%) underwent catheterization

and a Doppler echo examination[20]. In all cases simultaneous recordings were made of LV pressures and Doppler velocity curves and the following were calculated: maximum rate of pressure increase during isovolumic systolic (peak + dp/dt, maximum rate of pressure decrease (peak-dP/dt) and the time constant of pressure reduction during isovolumic relaxation (tau). The Tei index was found to be significantly correlated with all three variables, providing confirmation that it is a reliable measure of total LV function. The index was also found to be more sensitive in the evaluation of diastolic relaxation than parameters such as the deceleration time of the E wave (DT) and the E/A ratio, which showed a weaker correlation with peak-dP/dt and tau.

MYOCARDIAL PERFORMANCE INDEX AND CORONARY ARTERY DISEASE

The Tei Index was extensively evaluated by many authors in patients with asymptomatic coronary artery disease and in patients with myocardial infarction.

The Strong Heart Study - Rakesh K.Mishra et al, 1862 American Indian Participants free of Coronary (or) valvular disease (or) LV Systolic dysfunction were studied. Mean age was 59 ± 8 years, 66% were females, 48% had DM Type 2, 44% had SHT and 54% were obese[21]. All these patients base line LV function was Normal. MPI was done in all patients, to assess its prognostic value for incident cardiovascular events, including nonfatal stroke, CAD, CCF and Cardiovascular Death. There were no significant associations of MPI with SHT, Diabetic status, systolic (or) Diastolic BP, BMI, HbA1C, or LV Mass. MPI did not predict Fatal & nonfatal LV events at a mean follow up of 7.1 ± 2.2 years. In a population based sample of adults with high prevalence of DM 2, SHT, Obesity, but **without overt cardiovascular disease.** MPI has weak associations with clinical and physiological

determinants of cardiac function. Moreover, MPI does not provide prognostic information for CV events in this population. Though conceptually attractive as a global measure of cardiac function, MPI has limited utility in a high-risk population without clinical CV disease.

In a prospective study by **Maddury Jyotsna etal**, 63 patients with **chronic stable angina** without previous MI and with **Good LV Function** were studied[22]. 32 patients were without critical coronary stenosis by CAG, 31 patients had atleast > 70% occlusion in one vessel by CAG. There were no significant differences in IVCT, IVRT, ET and MPI. MPI was not informative in this study group.

MPI IN ACUTE MYOCARDIAL INFARCTION

In patients with Myocardial Infarction, systolic dysfunction results in a prolongation of the pre ejection (IVCT) and shortening of the ejection free (ET). Both Systolic and Diastolic Dysfunction result in abnormality in myocardial relaxation, which prolongs the relaxation period (IVRT). As a result there is **prolongation of IVCT, IVRT and shortening of ET**. So MPI increases and provides functional information.

MPI IN RELATION TO SITE OF MYOCARDIAL INFARCTION.

Patients with anterior wall MI has MPI values higher than in patients with Inferior wall MI. Patient with IWMI with RVMI has MPI more than patients with Isolated IWMI.

To define the degree of heart derangement in recent myocardial infarction (MI) occuring in different wall locations of myocardium, **Caccipuoti F et al** echocardiographically evaluated left ventricular volumes, ejection fraction, wall-motion score index, isovolumetric contraction

and relaxation time, ejection time, and the index of myocardial performance in 74 patients with MI[23]. Mean values of LV end-diastolic and end-systolic volumes and ejection fraction were nearly alike in all patients, where as wall motion score index of myocardial performance were clearly prolonged in those with anterior MI in comparison with the values recorded in patients with lateral or inferior MI.

MPI reflects severity of LV Dysfunction.

In a study by **karvounis et al**, they evaluated the applicability of the Doppler-derived myocardial performance index (MPI), in patients with acute myocardial infarction (AMI), whether this index reflects the severity of LV Dysfunction[24]. Post-AMI patients were compared with age and sex matched healthy subjects. Patients were evaluated within 24 hours of the AMI and 1 month thereafter. Patients were divided into group A (Killip Class I), and group B (Killip Class II-III). The LV ejection fraction (EF), transmitral E and A waves, E/A ratio, deceleration time (DT), isovolumic contraction time (IVCT), isovolumic relaxation time (IVRT), MPI, LV end-systolic and end-diastolic volume indices (ESVi and EDVi) and wall motion score index (WMSI) were evaluated. It was found that EF, ESVi, MPI and WMSI were significantly elevated at day 1 in patients with Killip Class II, III, it was found to persist even after 1 month. One-year mortality was significantly lower in group A patients. This study shows that the MPI, reliably indicated LV dysfunction post-AMI, significantly correlated with clinically determined functional class, and possibly has some prognostic implication.

MPI IN PATIENTS WITH ISOLATED DIASTOLIC DYSFUNCTION.

MPI values in patients with isolated diastolic dysfunction have a pseudo normalisation pattern. So the validity of Tei index in patients with isolated diastolic dysfunction was

evaluated in a study by **Bruch et al**[25]**.** This study is carried out to validate the Tei-Index in CAD patients with overall cardiac dysfunction or isolated diastolic Dysfunction. Sixty subjects were included who underwent left heart catheterization for invasive measurement of left ventricular end-diastolic pressure (LVEDP): 20 symptomatic CAD patients had Overall cardiac dysfunction (defined by a LV ejection fraction (EF) < 45% and a LVEDP ≥ 16 mm Hg, NYHA class 2.7 ±-0.4, OCD group), 29 symptomatic CAD patients had isolated diastolic dysfunction (defined by an EF > 45%, A normal end-diastolic diameter index and a LVEDP ≥ 16 mm Hg, NYHA Class 2.3 ± 0.4, IDD Group) and 11 asymptomatic control subjects (EF 65 ±- 9%, LVEDP 11 ± 4 mm Hg, CON group) were studied. The Tei-Index was easily and reproducibly measured in all study subjects. In the OCD Group, isovolumic contraction time was prolonged and ejection time was shortened in comparison to the CON group, resulting in a significantly increased Tei-Index. In the IDD group, isovolumic relaxation time was prolonged and isovolumic Contraction time was shortened in comparison to controls, resulting in a largely unchanged Tei-Index. Using a Tei-Index >0.49 as a cut-off, OCD patients were identified with a sensitivity of 96% and a specificity of 86%. This study concludes that the Tei-Index is a valid and readily derived indicator of global cardiac dysfunction in CAD patients with impaired systolic and diastolic LV performance. But the use of this index seems to be limited in CAD patients with primary diastolic dysfunction.

PSEUDONORMALISATION OF MPI.

With deteriorating Myocardial function (indicated by prolongation of IVCT, shortening of ET, and appearance of Restrictive filling pattern) an increased in MPI is prevented by a shortening of IVRT. The shortened IVRT time reflects, of course, an increase in left atrial pressure and not an improvement in relaxation. The lack of increase in MPI is therefore misleading. The behaviour of MPI can be accurately termed as pseudonormalisation, since it has the same pathophysiologic basis as pseudonormalisation of LV filling pattern.

PROGNOSTIC IMPLICATIONS OF MPI.

SHORT TERM PROGNOSIS

In a prospective study[26], MPI was done within one hour of hospitalisation of Acute Myocardial Infarction, and High MPI predicts in-hospital development of congestive cardiac failure. In a study by **Poulsen SH et al:** 64 patients with acute myocardial infarction (MI) admitted to the hospital MPI was done within 1 hour after their arrival to the hospital. In patients with MI and in-hospital congestive heart failure (CHF), the index was significantly higher compared with patients without CHF. In a multivariate regression analysis, the index > 0.45 was the strongest independent predictor of the development of CHF. This simply obtained non-geometric Doppler index, assessed in the early phase of MI, detected and graded left ventricular dysfunction and identified patients at risk for the development of CHF.

In a study[27] by **Jacob E Moller et al,** MPI performed within 24 hours of hospitalisation, on day 5, 1 and 3 months of MI in 125 consecutive patients. The index measured on day 1 correlated well with the change in end-diastolic volume index observed from day 1 to 3 months following AMI ($r = 0.66$, $p < 0.0001$). One-year survival in patients with Doppler index <0.63

was 89%, and 37% in patients with index **≥ 0.63**, ($p < 0.0001$). Multivariate analysis identified myocardial performance index ≥ 0.63 (relative risk 5.6, $p < 0.0001$), E-wave deceleration time < 140 ms (relative risk 2.7, $p = 0.008$) and Killip Class ≥ II (relative risk 4.0, $p=0.04$) to be independent predictors of cardiac death. Hence it was concluded that the Doppler echocardiographic myocardial performance index is a predictor of LV dilation and cardiac death after a first AMI. Another study[28] by **Poulsen SH et al:** Doppler index was measured in 60 consecutive patients with AMI and in 30 patients admitted to hospital with suspected but disproved AMI who served as controls. The patients were studied at days 1, 5, 90, and 360 after arrival in the coronary care unit. The index was significantly higher in patients with AMI than in control subjects at days 1 and 360. The index was significantly higher in patients who developed congestive heart failure or died compared with survivors who were free of congestive heart failure. Univariate analysis demonstrated that the Doppler index ≥ 0.60, deceleration time <140 ms, ejection fraction <40%, anterior wall MI, and age were significant predictors of outcome. Multivariate stepwise analysis showed that the index ≥ 0.60, deceleration time < 140 ms, and age were independent predictors of outcome.

LONGTERM PROGNOSIS

Szymanski P, et al evaluated the long-term prognostic value of MPI in 90 patients discharged from hospital after acute myocardial infarction (AMI)[29]. All the patients were followed for an average of 58 months. After multivariate Cox analysis, Tei index > 0.55 LV end-systolic volume > 65 ml, and mitral E wave deceleration time ≤145msec were the only independent predictors of cardiac events during the follow-up period. In a sub group of patients with Preserved LV Systolic function (ejection fraction > 0.40), MPI is the only

predictor of cardiac events.

MPI FOR ASSESSMENT OF LV OUTCOME AFTER PCI.

This study[30] was done by **M Kato et al,** to evaluate whether the myocardial performance index (MPI) can predict left ventricular functional outcome in patients with early recanalisation after anterior acute myocardial infarction (MI) and to determine when the index should be measured. 32 consecutive patients with their first anterior acute MI who had complete occlusion of left anterior descending coronary artery who underwent successful PCI within six hours of symptom onset in whom LV wall motion score index (A-WMSI), left ventricular end diastolic pressure (LVEDP), left ventricular ejection fraction (LVEF), and left ventricular end diastolic volume (LVEDV) were measured. It was found that there was a significant negative correlation between MPI on day 2 and the coronary diastolic deceleration time as well as a significant positive correlation with the coronary diastolic deceleration rate. MPI on day 2 was significantly correlated with the short and long term changes of A-WMSI and with the short term changes of LVEDP. Further more, MPI on day 2 was significantly correlated with the short and long term changes of LVEF and of LVEDV. From this, it can be concluded that Doppler derived MPI on day 2, representative of the early coronary micro vascular state, can predict the LV functional outcome after early successful recanalisation of a patients with first anterior acute MI.

In another study by **Sasao H, et al** evaluated the usefulness of the Tei index for assessment of infarct size and clinical outcome in patients with AMI treated by successful primary angioplasty[31]. 10 age-matched control subjects and 43 consecutive patients with first

AMI treated by successful primary angioplasty were analyzed. The Tei Index of the AMI patients was significantly greater than that of the control subjects. Also, the Tei Index has showed a significant positive correlation with peak creatine Kinase values and (99m) Tc tetrofosmin scores. Moreover, multiple logistic regression analysis showed that the Tei Index > 0.70 was the only significant explanatory factor for cardiac death or developed congestive heart failure. The Tei index combining systolic and diastolic myocardial performance reflects infarct size and might be a predictor of clinical outcome in patients with AMI treated by successful primary angioplasty.

MPI IN RELATION TO ANGIOGRAPHIC SEVERITY OF CORONARY ARTERY LESION.

In a study by **Kurtulu Ozdemir MD et al**, which was designed to find out the usefulness of peak myocardial systolic velocity (Sm) and MPI of RV measured by Pulse wave tissue Doppler imaging in assessing RV function[32]. 60 patients who experienced a first acute IWMI ± RVMI were taken for study. ECG showed IWMI + RVMI in 16 patients and others without RVMI. From echocardiographic A4C view, the peak myocardial systolic velocity, peak early diastolic velocity, ET, IVRT, IVCT of RV were measured at the level of tricuspid annulus using TDI then MPI was calculated. Echo done within 2nd day of admission. Coronary angiography and the Left ventriculography were performed to detect the Infarct Related Artery within 1st month after MI. Total and subtotal occlusion of the Coronary Artery supplying the asynergic field, as seen in the features of Left Ventriculography and angiographic presenting acute thrombosis or dissected plaque was accepted as the defining features for Infarct Related

Artery. The patients were divided into three groups according to the level of RCA lesion when IRA was RCA. The patients with the lesion proximal to Acute Marginal were defined as Group I, and those with lesions distal to the marginal branch of RCA were defined Group II. IRA in 27 patients was proximal RCA. RV Sm was observed to be significantly low in patients with RVMI and those in-group I compared to other groups. ($P< 0.001$). The sensitivity and specificity of Sm < 12 cm/s in diagnosing RVMI was 81%, 82% and in the diagnosis of proximal RCA as IRA, these values 63%, 88%. MPI was high in patients with RVMI compared to those who don't ($P < 0.001$). The sensitivity and specificity of MPI of > 0.70 in the diagnosis of RVMI was calculated as 94% and 80%. In the diagnosis of the proximal RCA as the IRA, these values were 78% and 91%. This study concluded that MPI might be a useful predictor of proximal RCA lesion.

In a study by **Sinan Dagdelan et al,** MPI was done in 82 patients to assess the importance of MPI in-patient with critical coronary artery disease[33]. This study showed that there was significant difference in IVRT, MPI, DT, in patients with critical coronary stenosis, when compared with those without critical stenosis. Hence this study concludes that MPI might be a useful parameter and an early indicator of LV dysfunction in patients with critical coronary artery disease.

Measurement of the Tei index is non-invasive and easily obtained, it does not require the presence of an echocardiographer with great experience and it does not materially prolong the time required for the examination. The calculation of the index is not based on a geometric model or on volume measurements; it is first and foremost a ratio of time intervals,

independent of ventricular geometry. It is also independent of blood pressure, heart rate and age and it appears to be of great prognostic value in many different clinical settings and it is suitable for follow up studies and is cost effective. Of course, the Tei index has its disadvantages and its use may present difficulties. For example, its precise measurement is infeasible in patients with atrial fibrillation, frequent ventricular ectopic stimuli, disturbances of intraventricular or atrio-ventricular conduction, a permanent pacemaker and in patients with severe aortic or mitral valve disease, severe pericardial effusion. Because these factors significantly influence the patterns of LV inflow and outflow.

MATERIALS AND METHODS

This study was conducted in 50 patients who were admitted to CCU, Department of Cardiology, Stanley medical college with acute myocardial infarction (STEMI).

SELECTION CRITERIA

Patients who were admitted to CCU, with first episode of acute myocardial infarction were included. The diagnosis of myocardial infarction was based on presence of any 2 of the following 3 criteria,

1. Typical precordial pain
2. ECG changes suggestive of MI (ST segment elevation of >0.1 mv in limb leads or > 0.2 mv in precordial leads)
3. Elevated cardiac enzymes.

The diagnosis of first episode of MI was determined if the previous ECG was normal or there was no history or symptoms suggestive of coronary disease.

EXCLUSION CRITERIA

The following groups of patients were excluded from the study. Patients with

1. Significant Valvular heart disease
2. Pericardial disease
3. Cardiomyopathies
4. Unstable angina
5. Significant tachy or bradyarrythmias.
6. On pace maker therapy

The entire patients who were included in the study were evaluated on the basis of Proforma, detailed history with special focus on chest pain duration and risk factors were

obtained. Patients were evaluated with a thorough echocardiographic analysis within 48 hrs of admission. Their treatment history and in hospital complications were noted.

TWO –dimensional and M-mode measurements were obtained with patients in left lateral position using an ALOKA SSD4000 phased array system equipped with tissue Doppler and harmonic imaging technology. Para sternal long and a short axis as well as apical four chamber, five chamber, and two chamber views were used for the evaluation of the functions of the left ventricle and the heart valves .LV dimension and fractional shortening (FS) of the left ventricle were calculated by using teicholtz formula. Ejection fraction was obtained by modified Simpson's method.

Pulsed-wave Doppler measurements of mitral inflow were obtained with the transducer on the four-chamber view with a 1-2mm Doppler sample volume was placed between the tips of the mitral leaflets during diastole. The left ventricular outflow velocity curve was recorded from the apical five-chamber view with the sample volume positioned just below the aortic valve.

Doppler velocities and time intervals were measured from mitral inflow and left ventricular outflow recordings. Isovolumetric relaxation time (IVRT) was the time interval from cessation of left ventricular outflow to onset of mitral inflow, ejection time was the time interval from the onset and cessation of left ventricular outflow, deceleration time (DT) was the time interval between the peak E velocity and the end of the early diastolic flow. Total systolic time interval was measured from the cessation of one mitral flow to the beginning of the following mitral inflow. Isovolumetric contracting time (IVCT) was calculated by subtracting

ET and IVRT from the total systolic time interval. MPI was calculated by using the formula MPI = (IVRT+IVCT)/ET.

Tissue Doppler echo was performed by activating the tissue Doppler function in the same machine. Images were obtained in the apical four chamber view with the filter setting were kept low and gains were adjusted at the minimal optimal level to minimize noise. 1.7 mm sample volumes were placed at both septal and lateral mitral annular site and systolic velocity (Sm) early and late diastolic velocities (Em or E', Am or A') were obtained and average values were taken. E/E' ratio was calculated.

Coronary angiogram done in the Siemens mobile unit cath lab in our hospital. Coronary angiogram done through right femoral approach and with judkins technique. Low osmolar nonionic contrast agent (omnipaque) was used. Multiple views are taken to make sure that all coronary segments are seen clearly without foreshortening or overlap. Quantitative analysis was done with a medical imaging system CMS analysis software.

Statistical Analysis.

Data's are expressed as mean value ± standard deviation (SD). Statistical significance was defined as $P < 0.05$.Statistical analysis was done using SPSS software system.

RESULTS AND ANALYSIS

50 consecutive patients admitted in our ICCU with Acute myocardial infarction were included in the study after obtaining informed consent. Among the 50 patients 35 (70%) were males and 15(30%) were females. Out of 35 male patients 14 had anterior, 9 had antero septal,

9 had inferior ± lateral or dorsal MI, and 3 had inferior with RV myocardial infarction. Among the 15 females 5 had anterior, 6 had anteroseptal, 2 had inferior and 1 had inferior with RV myocardial infarction.

They were thoroughly evaluated by 2D, M-Mode, Doppler and Tissue Doppler echocardiography, and coronary angiography. The results of the study were as follows,

AGE & MPI

AGE	N	MPI Mean	Std. Deviation	One way ANOVA F-test
36-45	12	.5875	.07250	F=0.38 P=0.63 Not significant
46-55	22	.6309	.09836	
56-65	16	.6106	.20818	
Total	50	.6140	.13747	

SEX and MPI

SEX	N	MPI Mean	Std. Deviation	Student independent t-test
Male	35	.6174	.13870	t=0.26 P=0.79 Not significant
Female	15	.6060	.13902	

SK and MPI

SK	N	MPI Mean	Std. Deviation	Student independent t-test
Yes	39	.6131	.12138	t=0.08 P=0.93 Not significant
No	11	.6173	.19132	

Killps Class and MPI

Killip	N	MPI Mean	Std. Deviation	Oneway ANOVA F-test
1	30	.5873	.10674	F=2.40 P=0.10 Not significant
2	17	.6371	.15361	
3	3	.7500	.25710	
Total	50	.6140	.13747	

EF & MPI

!Unexpected End of Formula	N	MPI Mean	Std. Deviation	Oneway ANOVA
<35	5	.8600	.20736	F=14.38 P=0.001 Significant
36-45	7	.6829	.07477	
46-55	26	.5896	.09237	
>55	12	.5242	.07329	
Total	50	.6140	.13747	

E/A and MPI

E/A	N	MPI Mean	Std. Deviation	One way ANOVA

<1	11	.6564	.19926	F=2.01
1-2	36	.5919	.11275	P=0.15
>2	3	.7233	.07371	Not significant
Total	50	.6140	.13747	

DT and MPI

DT	N	Mean	Std. Deviation	One way ANOVA F-test
<160	23	.6548	.16096	F=3.98
160-240	27	.5793	.10473	P=0.05
>240	0	0.0	0.0	Significant

IVRT and MPI

IVRT	N	Mean	Std. Deviation	One way ANOVA F-test
<70	4	.6675	.14080	F=0.87
70-90	23	.5883	.13159	P=0.47
>90	23	.6304	.14345	Not significant
Total	50	.6140	.13747	

E/E' and MPI

E/E'	N	Mean	Std. Deviation	One way ANOVA F-test

<8	17	.5947	.07706	
8-12	26	.6188	.17037	
>12	7	.6429	.12419	F=0.33 P=0.72
Total	50	.6140	.13747	Not significant

MPI in Proximal LAD lesions.

LAD stenosis	N	Mean	Std. Deviation	Oneway ANOVA F-test
50%	3	.5967	.01528	
70%	10	.6480	.09647	F=4.13
90%	7	.7829	.22925	P=0.01
Distal	15	.5940	.05889	Significant
Total	35	.6474	.13581	

MPI in Proximal LAD vs Distal LAD lesions.

LAD	N	Mean	Std. Deviation	Student independent t-test
Proximal	20	.6875	.16280	t=2.37 P=0.03 Significant
Distal	15	.5940	.05889	

MPI in Proximal LCX lesions.

LCX	N	MPI Mean	Std. Deviation	Student independent t-test

Proximal	4	.5520	.13231	t=0.62
Distal	7	.5040	.02881	P=0.44 Not significant
Total	11	.5360	.10973	

MPI in proximal RCA Lesion

RCA	N	MPI Mean	Std. Deviation	Student independent t-test
Proximal	3	.6600	.14422	t=2.60
Distal	1	.5050	.07937	P=0.05 significant

MPI in DVD

DVD	N	MPI Mean	Std. Deviation	Student t-test
Yes	27	.6541	.16080	t=2.44
No	23	.5670	.08514	P=0.02 Significant

MPI in TVD

TVD	N	MPI Mean	Std. Deviation	Student t-test
Yes	7	.6986	.21805	t=1.96 P=0.05 Significant
No	43	.6002	.11777	

DISCUSSION

MYOCARDIAL PERFORMANCE INDEX AND CLINICAL, ECHO

PARAMETERS IN MI PATIENTS*:*

Among the 50 patients studied, 35 were male and 15 were female patients. When we analyzed MPI in relation to parameters like age, sex, chest pain duration, and Thrombolytic state we found that there was not much difference statistically.

MPI and Killip class*:*

Among the 50patients 30 (60%) were Killip class I, 17(34%) belongs to Killip class II, and 3 (6%) Belongs to Killip class III. When we analyzed the killip class of the patients and its relation to MPI we found that MPI was significantly higher when the killip class of the patients increased, while it was 0.58±0.10 in class I, 0.63±0.15 in class II, and 0.75±0.25 in class III. Our findings were similar to those observed by **Karvonish et al,** in their study[24] they found that MPI was higher in killip class II & III compared to Killip class I (MPI of 0.68 Killip class II & III against 0.34 in killip class I). It is a well established fact that killip class correlates with possible MI outcomes, since MPI reflects the Killip class it can also be used for assessing post MI out come. Higher MPI is associated with poor outcomes.

MYOCARDIAL PERFORMANCE INDEX AND SYSTOLIC FUNCTION PARAMETERS*:*

LV DIMENSION AND VOLUME

We found in our study that there was statistically significant correlation between MPI

and LV dimension, volumes. With an increase in LV dimension &volumes there was an increase in MPI. Our findings were similar to that of **Lanine et al** finding, in their study they[34] found that MPI was significantly higher in patients, who developed heart failure, with increased LV dimension & decreased Ejection fraction.

EJECTION FRACTION

When we analyzed the relationship of Ejection fraction with Myocardial performance index we found that with decreasing level of EF, there was an increase in MPI, while it was 0.52±0.07 in patients with EF >55; 0.58±0.09 in patients with EF of 46-55; 0.68±0.07 in patients with EF of 36-45; and it was 0.86±0.20 in patients with EF<35.This statistically significant **inverse correlation of MPI with EF** implies that MPI has got good correlation with reduction in LV systolic function, similarly MPI was found to be correlated well with other systolic function parameter like FS also.

MPI AND DIASTOLIC FUNCTION PARAMETERS

When we analyzed the relationship of MPI with diastolic function parameters like E/A ratio, IVRT, DT, E/E' ratio. We found that MPI was significantly associated with abnormalities in all the above mentioned diastolic function parameters except E/A ratio.

MPI and E/A RATIO:

When analyzing E/A ratio with MPI we found that MPI was higher when the E/A ratio was less than 1or more than 2.While MPI was 0.65±0.19 in patients with E/A ratio of <1, it was 0.72±0.33 when E/A was >2, when E/A ratio was between 1-2the MPI value was

0.59±0.11.This also reflects the pseudonormalisation of MPI like E/A ratio. Although MPI was high when there was abnormality associated with E/A ratio but it does not attained statistical significance.

MPI and DT:

When we analyzed Deceleration time with myocardial performance index, MPI was significantly increased when there was abnormality in Deceleration time. MPI was 0.65±0.16 when DT was lesser than 160, when compared to 0.57±0.10 when DT was between 160-240.Our findings were similar to **Poulsen et al's** finding, they also found in their study[26] that MPI was significantly higher in patient's with Deceleration time of < 140 msec. They had concluded in their study that decreased Deceleration time and elevated MPI were independent predictors of clinical out come.

MPI and IVRT:

In our study patients with IVRT <70msec indicating restrictive filling pattern has a MPI of 0.66±0.14.compared to patients with impaired relaxation pattern, in whom IVRT >90msec has an MPI of 0.63±0.14.patients with IVRT 70-90msec has MPI 0.58±0.13.In patients with impaired relaxation and restrictive filling pattern MPI is significantly increased (p = 0.01)

MPI and E/E'

In many studies elevated E/E' ratio of > 15 is a good marker of increased LVEDP more than 20 mm Hg. In our study patients with E/E' ratio less than 8 had MPI 0.59 ± 0.07 and the MPI values for E/E' ratio 8-12 are 0.61 ± 0.17. In patients with increased E/E' ratio > 12 had high MPI value of 0.64 ± 0.12. Patients with high E/E' ratio has got high MPI values.

MPI AND ANGIOGRAPHIC

SEVERITY OF CORONARY ARTERY LESIONS

MPI IN ANTERIOR WALL MI.

Among the 50 patients studied, 35 had AWMI (or) ASMI suggesting a lesion in LAD territory. In these patients, 20 patients had hemodynamically significantly proximal LAD lesion (defined as atleast 50% diameter stenosis). 3 Patients had Proximal LAD Lesion of 50%, 10 patients had proximal LAD Lesion of 70%, and 7 patients had proximal LAD lesion of 90%; 15 patients had either non significant lesions in Proximal LAD, (or) Luminal irregularities in Proximal LAD (or) Lesions in other sites of LAD. (Other than Proximal LAD).

In patients with Proximal LAD Lesion with 50% Diameter stenosis had MPI 0.59 ±

0.15. In patients with 70% Diameter Stenosis in Proximal LAD had MPI 0.64 ± 0.09 and in patients with 90% Diameter Stenosis the mean MPI value was 0.78 ± 0.22. In patients with insignificant lesions or with Distal Lesions had MPI value of 0.59 ± 0.05. When compared to Hemodynamically significant proximal LAD Lesions with Distal LAD Lesions, patients with Proximal LAD Lesions has MPI value 0.68 ± 0.16 and patients with Distal LAD Lesions has MPI value of 0.59 ± 0.05. (P = 0.03).

MPI IN IWMI / RVMI

Among 15 patients with IWMI, 4 patients have associated RVMI. In these 4 patients 3 patients had Proximal RCA Lesion, MPI Value in patients with Proximal RCA Lesion was 0.66 ± 0.14, compared to non proximal lesions the MPI value was 0.50 ± 0.07. (P=0.05)

These findings correlate with study by **Kurtulu Ozdemir MD etal**, and in their Study[32] Angiogram done one month after Myocardial Infarction in patients with IWMI + RVMI showed MPI value of > 0.70 in patients with RVMI has severity 94% and specificity of 80% to predict Proximal RCA Lesion.

In patients with IWMI without RVMI we found lesion in Proximal LCX in 4 patients, in other patients non-proximal lesions found in RCA and LCX. In patients with Proximal LCX lesions MPI value was 0.55 ± 0.13 and in patients with Distal Lesions the MPI was 0.50 ± 0.02 even though the MPI value was high in Proximal Lesions it does not reach statistical significance.

MPI IN DOUBLE VESSEL DISEASE

Among the 50 patients we studied, 16 patients had Single Vessel Disease, 27 patients had Double Vessel disease and 7 patients had Triple Vessel Disease. Mean MPI value in patients with Double Vessel Disease is 0.65 ± 0.16 compared to patients with Single Vessel Disease in whom Mean MPI value is 0.56 ± 0.08 which is statistically significant (P=0.02).

MPI IN TRIPLE VESSEL DISEASE

Among the 7 patients with Triple Vessel Disease the Mean MPI value is 0.69±0.21 and compared to others 0.60 ± 0.11. Patients with Triple Vessel Disease have significantly higher MPI (P=0.05).

CONCLUSION

The following conclusions were derived from our study

- Myocardial performance index is significantly higher in anterior infarcts than in Inferior infarct patients.
- Myocardial performance index has got a good correlation with systolic as well as diastolic function parameters. While Myocardial performance index was found to have a significant inverse relationship with Ejection fraction, it was also found to have significant relationship with abnormalities in diastolic function parameters like Deceleration time, Isovolumic relaxation time and E/E' ratio.
- Myocardial performance index has got a significant positive correlation with Killip class.
- Myocardial performance index is significantly increased in patients with proximal LAD, proximal RCA lesions compared to distal lesions.
- Myocardial performance index is significantly elevated in Double Vessel Disease and Triple Vessel Disease compared to Single Vessel Disease.

BIBLIOGRAPHY

1. **Braunwald's** heart disease. 7th edition. 491-507
2. **Feigenbaum's echocardiography**. 6th edition. 437-487
3. **Nishimura R, Tajik AJ**, evaluation of diastolic filling of left ventricle in health in disease; Doppler echocardiography is the clinicians rosetta stone.JACC.1997; vol 30;page 8-18
4. The **Echo manual JK .OH.** 3rd edition. 120-142
5. **Dodge JT**, lumen diameter of normal human coronary arteries. Influences of age, sex, anatomic variations and left ventricular hypertrophy and dilatation. Circulation 19992,volume 86, 232
6. **HURST'S THE HEART**.11th edition. Page 481-541
7. **Uren Na**, Relation between myocardial blood flow and the severity of coronary artery stenosis, NEJM 1994.vol 330.page 1782.
8. **Sheehan F, Braunwald E, etal**; the effect of intravenous thrombolytic therapy on left ventricular function; A report on the tPA and SK from TIMI phase I trail. Circulation 1989.vol 72.page 812
9. **Gibson etal**; TIMI frame count; A quantitative method of assessing coronary Artery flow. Circulation 1996.vol 93.page 879-888
10. **Koerselman J etal**; coronary collaterals; an important and underexposed aspect of coronary artery disease. Circulation 2003;vol 107.page 2507
11. **Dewood MA etal**; prevalence of total coronary artery occlusion during the early hours of transmural myocardial infarction. NEJM 1980.vol 303.page 897
12. **Gash, AK, Spann, JF, et al;** Factors influencing reocclusion after thrombolysis for

acute myocardial infarction. Am J cardiol.1986. vol56. page175

13. **Weissler etal**; systolic time intervals in heart failure in man. Circulation 1968. vol 37. Page 149-159

14. **Mancini GBJ, etal**; the isovolumic index-a new non invasive approach to the assessment of left ventricular function in man.JACC.1982.vol 50.page 1401-1408

15. **Tei C, Ling L, Hodge D, etal**; new index of combined systolic &diastolic myocardial performance; a simple and reproducible measure of cardiac function; a study in normals and dilated cardiomyopathy; J OURNAL OF CARDIOLOGG, 1995.vol 26.page 357-366

16. **Harada k, Orino T, yasuoka K et al:** Tissue Doppler imaging of the left and right ventricle in normal children. Tohoku J Exp Med 2000; 191:21-29.

17. **Eto G, Ishili M, Tei C, Tsitsumi T, Akagi T, Kato H:** Assessment of global left ventricular function in normal children and in children with dilated cardiomyopathy. J Am Soc Echocardiogr 1999; 12:1058-1064.

18. **Moller J, Poulsen S, Egstrup K:** Effect of preload alternations on a new Doppler echocardiographic index of combined systolic and diastolic performance. J Am Soc Echocardiogr 1999; 135:1065-1072.

19. **Lars Lind M.D., The Doppler** derived myocardial performance Index is determined by both left ventricular systolic and diastolic function as well as by afterload and left ventricular mass. Echocardiography. Vol. 22 No.: 3,2005.

20. **Tel C, Nishimura R, Seward J, Tajik A:** Noninvasive Doppler-derived myocardial performance index: correlation with simultaneous measurements of cardiac catheterization measurements. Echocardiogr 1997; 10:169-178.

21. **Rakesh K.Mishra** Utility of myocardial performance index in a population with High prevalences of obesity, Diabetes and Hypertension. The strong heart study. Echocardiography Vol. 24: 4: 340-347 Apr. 2007.

22. **Maddury Jyostna etal; can** MPI detect significant Obstructive coronary artery disease, Chest Oct. 2003.

23. **Cacciapuoti F, Arciello A, Fiandra M et al:** Index of myocardial performance after early phase of myocardial infarction in relation to its location. J Am Soc Echocardiogr. 2004 Apr; 17(4): 345-9.

24. **Karvounis HI, Nouskas IG, Farmakis TM, et al:** Evaluation of a Doppler-derived index combining systolic and diastolic left ventricular function in acute myocardial infarction. Angiology. 2004 Jan-Feb; 55(1): 21-8.

25. **Bruch C, Schmermund A, Dagres N, Katz M, et al:** Tei-Index in coronary artery disease - validation in patients with overall cardiac and isolated diastolic dysfunction. Z Kardiol, 2002 Jun; 91(6): 472-80.

26. **Poulsen SH, Jensen SE, Tei C, et al: Value** of the Doppler index of myocardial performance in the early phase of acute Myocardial infarction. J Am Soc Echocardiogr. 2000 Aug; 13(8): 723-30.

27. **Jacob E. Moller, Ph.D., Kenneth Egstrup, DmSc, Lars Kober, Dmsc, et al:** Prognostic importance of systolic and diastolic function after acute myocardial infarction. Am Heart 2003; 145:147-53).

28. **Poulsen SH, Jensen SE, Nielsen JC, et al:** Serial changes and prognostic implications of a Doppler-derived index of combined left ventricular systolic and diastolic myocardial performance in acute myocardial infarction. Am J Cardiol 2000; 85:19-25.

29. **Szymanki P, Rezler J, Stec S, et al:** Long term prognostic value of an index of myocardial performance in patients with myocardial infarction. Clin Cardiol. 2002 Aug; 25(8): 378-83.

30. **M.Kato, K. Dote, S Sasaki, K Goto, H Takemoto, S Habara and D Hasegawa:** Myocardial performance index for assessment of left ventricular outcome in successfully recanalyzed anterior myocardial infarction. Heart 2005; 91; 583-588.

31. **Sasao H, Noda R, Hasegawa T, Endo A, Otimatsu H, Takada T :** Prognostic valve of the Tei index combining systolic and diastolic Myocardial performance in patients with acute myocardial infarction & Treated by successful primary angioplasty. Heart Vessels. 2004 Mar; 19(2)-68-74.

32**.** **Kurtulu Ozdemir MD et al**: New parameters in identification of RVMI and Proximal RCA lesion. Chest 2003. Vol. 123. Page 219-226.

33. **Sinan Dagdelen, M.D., Nevnihal Eren, M.D., Nuri Cagler, M.D.:** Importance of the Index of Myocardial performance in evaluation of left ventricular function. Echocardiography Vol. 19, no.4, 2002, 273-278.

34. **Steven j. lavine, M.D: Prediction** of Heart failure in Post Myocardial Infarction: Comparison of Ejection fraction, Trans mitral filling parameters, and the Index of Myocardial performance. ECHOCARDIOGRAPHY: Vol. 20, no.8, 2003. 691-701.

PROFORMA

MYOCARDIAL PERFORMANCE INDEX AS A PREDICTOR OF ANGIOGRAPHIC SEVERITY OF CORONARY ARTERY DISEASE IN ACUTE STEMI

NAME: **AGE:** **SEX:**

OCCUPATION: **C.D. NO:**

ADDRESS:

SOCIO ECONOMIC STATUS

RISK FACTOR PROFILE:

HYPERTENSION **SMOKING** **OBESITY (BMI)**

DIABETES **HYPER LYPIDEMIA** **PREVIOUS CAD**

FAMILY HISTORY OF CAD **personality**

CLINICAL PROFILE:

CHEST PAIN DURATION:

VITAL PARAMETERS: PR: **BP:**

KILLIP CLASS:

INFARCT TERRITORY:

THROMBOLYTIC STATUS:

DRUGS USED:

COMPLICATION

CHF ARRYTHMIAS PIA DEATH

INVESTIGATION:

BLOOD SUGAR: CARDIAC ENZYMES:

ECG:

CHEST PA:

ECHO CARDIOGRAPHY:

SYSTOLIC FUNCTION: LV DIMENSION: LVDd: LVDs:

EDV: ESV: EF:

FS: RWMA:

DIASTOLCI FUNCTION:

TRANS MITRAL FLOW PATTERN: E A E/A RATIO:

DT IVRT

PULMONARY VENOUS FLOW PATTERN: S/D RATIO

PV a VELOCITY

TISSUE DOPPLER E' A' E/E' RATIO

SYSTOLIC AND DIASTOLIC FUNCTION:

TEI INDEX: MEASURE A (END TO BEGINNING OF MITRAL FLOW)

MEASUREMENT B (ET)

(A-B) / B

IVRT IVCT

CORONARY ANGIOGRAM: LMCA:

LAD:

LCX:

RCA:

GLOSSARY

STEMI:	ST ELEVATION MYOCARDIAL INFARCTION
LVDd:	LEFT VENTRICULAR INTERNAL DIMENSION IN DIASTOLE
LVDs:	LEFT VENTRICULAR INTERNAL DIMENSION IN SYSTOLE
CHD:	CORONARY HEART DISEASE
DM:	DIABETES MELLITUS
LVH:	LEFT VENTRICULAR HYPERTROPHY
MI:	MYOCARDIAL INFARCTION
EF:	EJECTION FRACTION
EDV:	END-DIASTOLIC VOLUME
ESV:	END-SYSTOLIC VOLUME
LVEF:	LEFT VENTRICULAR EJECTION FRACTION
ESPVR:	END SYSTOLIC PRESSURE VOLUME RELATIONSHIP
IVCT:	ISOVOLUMETRIC CONTRACTION TIME
RVET:	RIGHT VENTRICULAR EJECTION TIME
TDI:	TISSUE DOPPLER IMAGING
DT:	DECELARATION TIME
NYHA:	NEWYORK HEART ASSOCIATION
AMI:	ACUTE MYOCARDIAL INFARCTION
IVRT:	ISOVOLUMIC RELAXATION TIME
LAD:	LEFT ANTERIOR DESCENDING ARTERY
LCX:	LEFT CIRCUMFLEX ARTERY
RCA:	RIGHT CORONARY ARTERY
LMCA:	LEFT MAIN CORONARY ARTERY